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CURRENT ANATOMY, PHYSIOLOGY, AND PATHOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.

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BULBAR PARALYSIS.

The remarkable activity displayed in the search after pathological knowledge has recently received apt elucidation by the almost simultaneous publication, from two different observers, of cases of apparently, judging from the symptoms, bulbar paralysis, but on post-mortem and microscopical examination found to be bearing of no close relationship to this part. The case of Hoppe, observed in Berlin and published in this country (Med. Rec., April 30, 1892), gives the details of three other cases besides his own, observed respectively by Wilks, Oppenheim, and Eisenlohr, and to these can now be added the case published by Senator (Neurolog. Central., March 15, 1892).

The remarkably loose way in which the name bulbar paralysis has been handled is a matter to be regretted; and now that we are seemingly on the brink of having a disease with apparently the same complex of symptoms as paralysis depending on bulbar lesion, it behooves us to have a clear and thorough comprehension of the leison productive of this disease in mind, when we make use of the term. Frequently we see writers using the term labio-glosso-laryngeal paralysis as synonomous with bulbar paralysis, and this is to be disparaged, seeing that the symptoms of bulbar paralysis may result from many different anatomical lesions, as the symptoms of disease of the spinal cord do, and the time may not be far distant when we shall be able to clinically differentiate between at least some of these affections.

Bastian states that bulbar paralysis may be dependent on (1) Traumatic causes. (2) Rupture of blood-vessels within the substance of the bulb. (3) Occlusion of blood-vessel supplying the bulb (leading to foci of softening). (4) Chronic meningitis. (5) Tumors originating in the bulbs or its meninges. (6) Disseminated sclerosis. (7) Degenerative changes in the motor ganglion cells (labio-glosso-laryngeal paralysis). To these apparently we must add another to include these cases of which we have spoken above, for although they are few in number it will be shortly remarked that other and like examples will be seen now that attention has been called to it. And whether to call it a general neurosis, as Oppenheim has done, or to believe that satisfactory proof of the lesion will yet be found somewhere in the motorial areas, will of course depend upon individual ideas.

Senator's case in brief is that of a laboring man, forty-one years old, who was received into the hospital on account of paralysis and dumbness. Twenty years before he had a sore on the penis with attending inguinal gland infiltration. About two months previous to his admittance he took cold and suffered somewhat from a cough, which afterward became more severe, but which did not however cause him to give up his work. About this time his wife began to notice that his speech had a marked nasal twang. A few days previous to his admittance he had some trouble in the night in swallowing and speaking; the tongue was very immovable and the jaws could not be widely separated; the facial expression was somewhat staring and the eyes very prominent; later on he became very restless, and the next day had loss of motion in right arm, which prevented him from writing his wishes as he had previously done. When he walks he is inclined to walk toward the left and drags his right leg after him somewhat.

He understands what is said to him, but cannot utter a tone or sound. He has some elevation of temperature and acceleration of pulse and respiration. The mobility of the right arm is very slight, in extension and supination of the forearm he cannot extend the fingers, the mobility in the right leg being considerably better. Plantar and patellar reflexes quick, right foot clonus more than left, and both cremaster reflexes alike. Bladder and rectum normal, no fibrillary twitchings in the affected muscles. Paralysis of the trunks and branches of both facial nerves, inability to raise or close the eyelids, or to wrinkle forehead, mouth constantly open, very little mobility of the tongue which, however, presents no atrophy or fibrillary twitchings. Sensation for pain, taste, temperature, position and location seemed to be unimpaired. Irritation of the conjunctival mucous membrane causes contraction of the eyelids. Sight, taste, hearing all normal. Feeding is painful, and fluids have to be poured into the mouth, or the stomach-tube used. A left pleural effusion from which he suffered had to be aspirated. Electrical examination of the muscles of the face showed that the left side of the face required a stronger current to cause contraction than did the right, and the contractions were lightning-like quick. The mechanical irritability of the facial muscles is very much lessened. At this time the restlessness of the patient increased and his memory was very deficient. From this time on for some days the patient seemed to have some returning muscular ability in the face and throat muscles, so that he was able to open the mouth better and bring out a very indefinite tone; his restlessness, however, continued unabated, so that it was necessary to resort to anodynes. Later on it was observed that it was possible for him to say one word, "ja," which, however, requires no articulation; and a remarkable change now also observed was that even a slight mechanical irritability of the chin and lips was manifested by an increased contraction of the muscles of these parts, the electrical conditions remaining as before. A relapse followed shortly, and the patient died with these paralytic symptoms greatly aggravated, from the complicating pulmonary trouble. This was after six months of illness, and four months after the symptoms of paralysis first showed themselves. The autopsy revealed left pleural adhesions, an encapsulated empyema of about the size of the fist, in the left upper lobe a cavity about two cm. in diameter, and extensive per-bronchial tubercles. Heart normal, mucous membrane of larynx pale and œdematous, and the vocal cords covered with a slimy exudation. Skull cap, normal; dura, hyperæmic; pia nonadherent, brain surface moist and shining, and on cut sections could be seen numerous small and large spots of recent hemorrhage; the remainder of the great and lesser brain was apparently normal, with the possible exception that the blood-vessels on cross-section in the internal capsule and the lenticular nucleus and external capsule seemed to be rather farther apart than normal. In the ventricles a considerable clear fluid was found. The pons, medulla, and trunks of the facial and pneumogastric and hypoglossal nerves were scrupously examined and absolutely nothing abnormal found. The muscles of the tongue showed very little change, as did those of the larynx, perhaps here and there a small collection of pigment or a few fibres undergoing fatty degeneration.

The case reported by Hoppe is very similar to the above, with the exception that the latter developed symptoms much more slowly, and after a period of about six months had a period of seeming alleviation of symptoms which lasted for a few months. This did not occur in the case reported by Senator, or at least if there was any amelioration of the symptoms at the time when the patient became able to make a tone for a few days, it was extremely transitory. Then again in the case reported by the latter there was more or less partial hemiplegia, in so much as the right arm and to a less extent the leg was disabled, while in Hoppe's case there was merely some weakness of the extremities. The microscopical examination of the central nervous system and the peripheral nerves gave just as little information as regards the lesion of the disease in the latter case as it did in the former, in fact by most careful examination the results were entirely negative.

Hoppe, in order to show the similarity in the course and development of the disease between his own case and those reported by the observers mentioned above, gives a short review of those cases and a table of comparison; this shows that the disease first manifested itself by some disturbance of speech, weakness of the muscles of mastication, more or less ptosis or strabismus, and generally weakness of the extremities; it shows also that in each case the course was chronic, lasting in each case considerably more than a year, and interrupted in its development by remissions. There was involvement of the functions of the oculo-motorius, motric fifth, superior facial, inferior facial, glosso-pharyngeal and pneumogastric nerves in each case to a very great extent. The hypoglosal in each case remained free. The electrical reactions showed no evidence of degenerative atrophy, and death in every case ensued from paralysis of the circulation and respiration. This was the case in Senator's patient also, for although that case presented some symptoms which cannot be reconciled with the symptoms found in the histories collected by Hoppe, it is to be remembered that many of these symptoms were the direct concomitants of the extensive sero-purulent pleurisy from which the patient suffered during the course of the paralysis. Hoppe says that this complex of symptoms can be differentiated from the atrophic form of bulbar paralysis (type Duchenne), from acute bulbar paralysis and from that condition known as pseudo-bulbar paralysis. This is quite possibly true regarding the first two mentioned, but as regards the last, viz., pseudo-bulbar paralysis, it is not so apparent. As he says, it may be differentiated by the mode of its development, its course and duration, and especially by the absence of any pathological changes after death. In most of these cases reported, the remissions, the chronicity, the unimpairment of nutrition in the tongue, the absence of any change in the electrical irritability, and the persistence of reflex action were all prominent, and this is most always the case in pseudo-bulbar paralysis. In pseudo-bulbar paralysis ordinarily there are two attacks, generally first attacking one side and then the other, and the second attack is generally followed by the paralysis of the lips, tongue, and pharynx; and although it is not clearly evident that there were two attacks in the cases reported by all these observers, in some of them, as in Eisenlohr's case, this was apparently so, where weakness of the lower extremities and ptosis took precedence of any symptoms pointing to bulbar implication; this was likewise so in the case reported by Oppenheim. The differentiation from pseudo-bulbar paralysis is not therefore so evident, nor is it easily made. The one factor that is undisputably present in these (4) cases commented on by Hoppe and in the case of Senator's is the absolute want of pathological change in central or peripheral nervous system on which the symptoms could be dependent; for in Hoppe's case, at least, the examination was thoroughly and exhaustively made and comprehended the entire central nervous system and the peripheral nerves.

Of course, the interesting point in the consideration of these cases hinges around the question, Are we to add a new disease, with a sufficiently well-marked symptomic entity to entitle it to a separate nomenclature to our present category? Oppenheim, following out a course of iniatitive, seemingly suggested by Charcot in discussing the history, a somewhat analagous condition would have us believe that the symptoms of his case and of the others spoken of are dependent on a neurosis, or rather that this conclusion is forced upon us as no other causation is apparent.

Hoppe tentatively suggests that perhaps the paralysis may be the result of ptomaine or toxic-albumen poisoning, and thinks that possibly the peri-bronchial tubercular nodules found in the case reported by him may stand in the light of a causative influence in developing these poisonous factors. Although such evidence of tubercular causation was not found in but three cases referred to by him, they were found in Senator's case. Of course this would be putting these cases under the heading of multiple neuritis; for although Hoppe says, in his opinion, the cortical centres of the brain may become affected from such a toxic product so as to cause a complex of symptoms whose main characteristic is a slowly progressing paresis, this is extremely improbable, for many reasons. In the first place, there is no initiative, either theoretical, clinical, or experimental, for making such a statement; and secondly, we know from both experimental and clinical observations that toxic products when in the blood, whether they are generated within the body or taken in from without, have a marked predilection for the peripheral nerves, and thus destructive influence becomes manifest here to such an extent as to cause death without causing any change whatever in the cortical substance. Of course, one can say that it may cause changes in the cortex, which we are as yet, by reason of our immature methods of treating the tissues for examination, unable to detect; but the advancement of such a hypothesis as an explanation of the pernicious effect of a toxic product is wholly unnecessary.

This does not apply to another suggestion made by Hoppe, in which he says: "I would like to call attention to the possibility that the cause of the disease may be a pathological change in the cortex of the brain which has escaped our attention." This idea deserves much more recognition than the others, for it is well known that great difficulty or loss of power of articulating, together with difficulty in swallowing, with or without some accompanying paresis of the limbs, may be occasioned by symmetrical bilateral lesions in the cortex of the cerebral hemispheres, occupying the posterior extremity of the third frontal and the inferior extremity of the ascending frontal convolutions; or also by a double implication of the efferent fibres from these convolutions when on their way toward the genu of the internal capsule. Such a lesion would not account for all the symptoms found in these cases, and particularly would it be found lacking in causing the mode of death by which these cases all terminated, viz., paralysis of respiration and circulation; for in such a lesion respiration and the action of the heart are but very little interfered with. A lesion, therefore, to cause all the symptoms recorded, would have to be rather extensive in its dissemination, and in these (5) cases there was evidence of mental aberration but in one, and that rather slight, it would appear that such a lesion would be removed entirely from that part of the cortical substrata which is the origin or home of intelligence.

Whether the case reported by Senator may be considered entirely in the same category as Hoppe's case and the others mentioned by him is questionable, as there was apparently some evidence of hemiplegia before or during the appearance of the bulbar symptoms, which may in part account for the latter.

The publication of such cases will at least stimulate us to be on the careful lookout for anatomical evidence of our cases of bulbar paralysis, and in this way tend to a solution of the apparent enigma. It also brings to remark on the many investigations that are being made to bring forth a thoroughly reliable stain for the cortical substance.

Our paucity of knowledge concerning the finer structure of the central nervous system, and the unsatisfactory results obtained with the ordinary staining methods, is the probable apology of the seeming rivalry existing between workers in histology, as to who can bring forth the greatest and most successful number of stains or to better those already existing. Some time since Berkley, of Baltimore (Johns Hopkins Hospital Bull., No. 13, 1891), suggested a method, seemingly based on Friedmann's method, and on Kaes' modification of Wolter's stain, and at the same time posessing some of the qualities that have served to place Ramon y Cajal's method so high in the estimation of scientists.

The method described as Berkley is briefly as follows: Pieces of tissue which must not be larger than 2.5 mm. in thickness, come for twenty-four to thirty hours in Flemming's solution, at a temperature of about 25° C., and then are at once placed in absolute alcohol without having been washed, and here they remain for twenty-four hours, the alcohol having been changed once in the meanwhile. Then if the pieces are sufficiently hardened they are laid in celluoidin for from twelve to twenty-four hours, and after that are ready for cutting, and this should be done with extreme care in order not to have the specimens too thick. After cutting, they are washed in water and then placed in a saturated solution of acetate of copper, where they remain over night ordinarily, but the process may be hastened by raising the temperature to 35–40 degrees for a half hour or so. After the coppering, the specimens are washed quickly in water, and then placed at once in the hæmotoxylin solution, which is prepared by taking 50 c.c. of distilled water, which is boiled thoroughly for a few minutes and then has added to it 2 c.c.m. of a saturated solution of lithium carbonicum, and then of a 10 per cent. alcoholic solution of hæmatoxylin, about 1.5 to 2 c.c.m. are added, then heated for a minute longer, the flask corked and allowed to cool, when it may be ready for use; but allowing it to stand for some hours is attended with better results. The time necessary for the specimens to remain in the hæmatoxylin varies from one to three minutes, then they are removed, thoroughly washed in water, dehydrated in alcohol, cleared in oil of bergamot, and mounted in xylol balsam.

The author remarks that the differentiation between the white and the gray substance is much less clearly marked than in preparations stained by Weigert's method; but with his osmium hæmotoxylin-copper method, with a little care and experience, the fibres, cells, and even the glia, will be stained, and the details of each well marked.

In the "N. Y. Medical Record" for March 12, 1892, this same author gives the results of some of his experiences with this stain on the cortex of the dog and the human being. In considering the cortex, he divides the medullated tubes of the gray layers into three principal systems—the radial, the inter-cellular, and the tangential or sub-pial fibres. The fibres of the radial layer passing out from the sub-cortical white matter in two different ways, one at an acute, the other at an obtuse angle, the first passing well into the intercellular layer without giving off any off-shoots, and the second beginning to give off off-shoots about opposite the fourth cellular layer, each off-shoot being directed toward a pyramidal cell and probably not going to one cell alone, but to two or more cells, and the impulses from several cells may not in this way be dependent for their transmission on the integrity of one fibre alone. It was not possible to trace each fibre to its cell, but a sufficient number were traced to state authoratively that such is the termination of the radial fibres. The fibres in the inter-cellular network are smaller than those of the tangential band, but there is considerable variation in their calibre. They have along their course regular swellings of the medullary sheaths, and it is from these that branching occurs. It is possible that these medullary nodes have a definite physiological meaning, as they are constant in perfectly hardened tissues that have been taken from the living animal and hardened in Flemming's fixation fluid. And it is the opinion of B. that they correspond to the nodes of Ranvier. The question of the branching of the fibres has not been in so unsettled a state as has the question of their anastomosing. Most anatomists are willing to concede the former, but this is by no means so of the latter. In a few instances B. says he has been able to determine the anastomosis with certainty, and he gives a very meagre figure in support of the anastomosis as seen.

He is able to corroborate the opinion of anatomists in general as regards the fact of the absence of non-medullated fibres, as he says, throughout the whole cortex no non-medullated fibres of any length were found to exist; so far as could be definitely determined, as soon as the protoplasmic character of the nerve-cell process is lost, the medullated character of the nerve fibre begins. It would, therefore, seem that the axis cylinder was immediately covered with an isolating envelope as soon as it had separated itself from the cellular protoplasm.

If this stain gives as good results in the hands of Dr. Berkley's co-workers as it has apparently done in his we have reason to congratulate ourselves on having taken a step in advance in regards to solving the histological structure of the cortex; the stains that we have at present have their greatest objections in their uncertainty, such as Golgi's and Ramon y Cajal on the one hand, and the method of preparation for the hæmatoxylin stains on the other; for it is forced upon us to believe that if we are to make serious strides forward in determining the lesions of the many so-called functional diseases which many believe to have their seat in structures of protoplasmic nature, it must be done by some method which does not entail its treatment by the potassium salts for weeks and even months which cannot but cause a partial obscuration of the conditions found in the recent state. Therefore a stain which seems to embody some of the virtues of the hæmatoxylin and the Cajal stain surely deserves a very extensive trial.

A NEW METHOD OF PREPARING BRAINS FOR PRESERVATION.

The preparation of brains for anatomical class-room demonstration has been rather unsatisfactory, and until the present time the method of treating them in a ten to twenty per cent. solution of nitric acid has been about the most satisfactory way, but this causes such shrinkage and alteration in the normal relation that the idea received from its study is generally not a satisfactory one, and must needs be greatly supplemented and cleared up by the study of the fresh brain. Recently Dr. L. Steida has made known a method by which the brain may be prepared in toto or in sections, showing normal relations very little altered (Neurolog. Cetralb., March 1, 1892). The method of preparation is very simple: The specimen is first immersed in a concentrated solution of chloride of zinc, sufficient of the liquid being used to allow the specimen to float. After remaining twenty-four hours in this, the specimen is removed, and although it is still soft and pliable, it is found to be possessed of sufficient toughness and consistency so as to allow the pia to be easily removed and without disturbing the relations of the convolutions or injuring the cortex. The brain, either in its entirety or after separation into parts, comes next, in a ninety-six per cent. solution of alcohol, where it remains for two or three weeks; and in order to expedite the process of hardening, it is advisable to renew the alcohol occasionally, say every five to six days. The entire brain or portions of it is now placed in turpentine, where it must remain from two to four weeks, depending on the quality of the turpentine and the extent and thoroughness of hydration caused by the alcohol; for if the brain be free from water when it is placed in turpentine the process will go on very rapidly. In summer the time required will be shorter than in winter, and it may also be hastened by the use of a small incubator; but great care is necessary to prevent shrinkage. By means of the turpentine the specimen becomes again soft but transparent, and it has acquired a brownish color, varying in tint with the kind of turpentine used. For the last part of the process it comes in oil varnish, where it remains for two weeks. Thereafter it is removed and allowed to stand for two weeks, or a shorter period, at the temperature of the room until it is dry and no longer oily to the touch. When complete, the specimen is of rather a pretty brown color, very little shrunken, and in good shape for study and demonstration.